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 NEWS  2  AUG 10  Time limit for inactive STN sessions doubles to 40
                  minutes
 NEWS  3  AUG 18  COMPENDEX indexing changed for the Corporate Source
                  (CS) field
 NEWS  4  AUG 24  ENCOMPLIT/ENCOMPLIT2 reloaded and enhanced
 NEWS  5  AUG 24  CA/CAplus enhanced with legal status information for
                  U.S. patents
 NEWS  6  SEP 09  50 Millionth Unique Chemical Substance Recorded in
                  CAS REGISTRY
 NEWS  7  SEP 11  WPIDS, WPINDEX, and WPIX now include Japanese FTERM
                  thesaurus
 NEWS  8  OCT 21  Derwent World Patents Index Coverage of Indian and
                  Taiwanese Content Expanded
 NEWS  9  OCT 21  Derwent World Patents Index enhanced with human
                  translated claims for Chinese Applications and
                  Utility Models

 NEWS EXPRESS MAY 26 09 CURRENT WINDOWS VERSION IS V8.4,
              AND CURRENT DISCOVER FILE IS DATED 06 APRIL 2009.

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DICTIONARY FILE UPDATES: 22 OCT 2009 HIGHEST RN 1189642-14-5

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=> s thalidomide
L1   29 THALIDOMIDE

=> file medline embase biosis
COST IN U.S. DOLLARS   SINCE FILE   TOTAL
                                                        ENTRY   SESSION
FULL ESTIMATED COST   5.83   6.05

FILE 'MEDLINE' ENTERED AT 15:42:01 ON 23 OCT 2009

FILE 'EMBASE' ENTERED AT 15:42:01 ON 23 OCT 2009
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FILE 'BIOSIS' ENTERED AT 15:42:01 ON 23 OCT 2009
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=> s l1
L2   20958 L1

=> s "idiopathic pulmonary fibrosis"
L3   6721 "IDIOPATHIC PULMONARY FIBROSIS"

=> s l2 and l3
L4   15 L2 AND L3

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5   10 DUP REM L4 (5 DUPLICATES REMOVED)

=> d l5 1-10 ibib abs

L5   ANSWER 1 OF 10 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights
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ACCESSION NUMBER: 2009441283 EMBASE
TITLE: Update in idiopathic pulmonary
                    fibrosis.
AUTHOR: Frankel, Stephen K.; Schwarz, Marvin I.
CORPORATE SOURCE: Division of Pulmonary Sciences and Critical Care Medicine,
                    University of Colorado Denver and Health Sciences Center,
                    Denver, CO, United States. frankels@NJHealth.org

AUTHOR: Frankel, Stephen K.
CORPORATE SOURCE: Department of Medicine, National Jewish Health G-012, 1400 Jackson Street, Denver, CO 80206, United States. frankels@NJHealth.org
AUTHOR: Frankel, S. K., Prof. (correspondence)
CORPORATE SOURCE: Department of Medicine, National Jewish Health G-012, 1400 Jackson Street, Denver, CO 80206, United States. frankels@NJHealth.org
SOURCE: Current Opinion in Pulmonary Medicine, (September 2009) Vol. 15, No. 5, pp. 463-469.
Refs: 39
ISSN: 1070-5287; E-ISSN: 1531-6971 CODEN: COPMFY
PUBLISHER: Lippincott Williams and Wilkins, 530 Walnut Street, Philadelphia, PA 19106-3621, United States.
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
017 Public Health, Social Medicine and Epidemiology
019 Rehabilitation and Physical Medicine
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 22 Sep 2009
Last Updated on STN: 22 Sep 2009

AB Purpose of review: This review aims to highlight and place in context recent advances in and insights into the natural history, diagnosis, and management of idiopathic pulmonary fibrosis (IPF). RECENT FINDINGS: Although the diagnosis of IPF remains challenging, an evolution in systems of practice and advancing technologies are steadily improving diagnostic accuracy. The identification of concomitant pulmonary hypertension as well as acute exacerbations of the underlying disease have taken on increasing importance in the natural history of IPF. Similarly, the management of IPF remains challenging, and although a number of recent trials of novel investigational agents for the treatment of IPF yielded negative results, at least one of these trials showed significant benefit suggesting progress in the treatment of this disease. SUMMARY: Although IPF remains a diagnostic and therapeutic challenge to even the most experienced of clinicians, our knowledge of the natural history of the disease, diagnostic accuracy, and therapeutic approach continue to advance.

L5 ANSWER 2 OF 10 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2009251228 EMBASE
TITLE: Advanced parenchymal lung disease: Quality of life and palliative care.
AUTHOR: Gilbert, Christopher R.
CORPORATE SOURCE: Division of Pulmonary and Critical Care Medicine, Department of Medicine, Thomas Jefferson University Hospital, Philadelphia, PA, United States.
AUTHOR: Smith, Cecilia M. (correspondence)
CORPORATE SOURCE: Department of Medicine, The Reading Hospital and Medical Center, West Reading, PA, United States. smithcm@readinghospital.org
SOURCE: Mount Sinai Journal of Medicine, (2009) Vol. 76, No. 1, pp. 63-70.
Refs: 63
ISSN: 0027-2507; E-ISSN: 1931-7581 CODEN: MSJMAZ
PUBLISHER: John Wiley and Sons Inc., 111 River Street, Hoboken, NJ 07030-5774, United States.

COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
017 Public Health, Social Medicine and Epidemiology
019 Rehabilitation and Physical Medicine
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 18 Jun 2009
Last Updated on STN: 18 Jun 2009

AB Advanced restrictive lung diseases remain a challenge for both the clinician and patient alike. Because there are few available treatment options that prolong survival for patients with diseases such as idiopathic pulmonary fibrosis, improvement in quality of life and palliation of significant symptoms become realistic treatment goals. Several validated instruments that assess quality of life and health-related quality of life have demonstrated the dramatic impact that lung disease has on patients. Quality-of-life assessments of patients with interstitial lung disease have commonly cited respiratory complaints as problematic, but other distressing symptoms often not addressed include fear, social isolation, anxiety, and depression. Not only do respiratory symptoms limit this patient population, but the awareness of decreased independence and ability for social participation also has an impact on the quality of life. Some patients describe a deepened spiritual well-being during their disease process; however, many patients' mental health suffers with experiences of fear, worry, anxiety, and panic. Many patients express desire for more attention to end-of-life issues from their physicians. Fears of worsening symptoms and suffocation exist with an expressed desire by most to die peacefully with symptom control. Interventions to improve quality of life are largely directed at symptom control. Pharmacologic and nonpharmacologic interventions have been helpful in relieving dyspnea. Studies have demonstrated that the use of supplemental oxygen in the face of advancing hypoxemia can have both positive and negative effects on quality of life. Patients using nasal prongs describe feelings of self-consciousness, embarrassment, and social withdrawal. Pulmonary rehabilitation is recommended, with some studies noting increased quality-of-life scores and decreased sensations of dyspnea. Sleep deprivation and poor sleep quality also have a negative impact on quality of life. Recognition and correction of nocturnal hypoxemia and other sleep disturbances should enhance quality of life in patients with restrictive lung disease; however, there is currently no evidence to support this claim. End-of-life care needs more attention by clinicians in the decision-making and preparatory phase. Physicians need to maintain their focus on quality-of-life issues as medical management shifts from curative therapies to comfort management therapies. Palliative care and hospice appear to be underused in patients with advanced diseases other than cancer. Because the only curative option for some end-stage restrictive lung diseases is lung transplantation, if transplantation is not an option, palliation of symptoms and hospice care may offer patients and families the opportunity to die with dignity and comfort. .COPYRGT. 2009 Mount Sinai School of Medicine.

L5 ANSWER 3 OF 10 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2008482983 MEDLINE
DOCUMENT NUMBER: PubMed ID: 18663075
TITLE: Thalidomide inhibits the intractable cough of idiopathic pulmonary fibrosis.
AUTHOR: Horton M R; Danoff S K; Lechtzin N
SOURCE: Thorax, (2008 Aug) Vol. 63, No. 8, pp. 749.
Journal code: 0417353. E-ISSN: 1468-3296.
PUB. COUNTRY: England: United Kingdom

DOCUMENT TYPE: (CLINICAL TRIAL, PHASE II)
Letter
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(CLINICAL TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200808
ENTRY DATE: Entered STN: 30 Jul 2008
Last Updated on STN: 26 Aug 2008
Entered Medline: 25 Aug 2008

L5 ANSWER 4 OF 10 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2008512890 EMBASE
TITLE: Potential of imatinib mesylate as a novel treatment for pulmonary fibrosis.
AUTHOR: Chhina, Mantej; Grant, Geraldine
CORPORATE SOURCE: Center for Biomedical Genomics, George Mason University, 10900 University Boulevard 109, Manassas, VA 20110, United States. ggrant1@gmu.edu; mchhina@gmu.edu
AUTHOR: Shlobin, Oksana A.; Nathan, Steven D. (correspondence)
CORPORATE SOURCE: Inova Lung Transplant Program, Inova Fairfax Hospital, 3300 Gallows Road, Falls Church, VA 22042, United States. steven.nathan@inova.org; oksana.shlobin@inova.org
SOURCE: Expert Review of Respiratory Medicine, (August 2008) Vol. 2, No. 4, pp. 419-431.
Refs: 85
ISSN: 1747-6348
PUBLISHER: Expert Reviews Ltd., 2 Albert Place, London, N3 1QB, United Kingdom.
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
015 Chest Diseases, Thoracic Surgery and Tuberculosis
017 Public Health, Social Medicine and Epidemiology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 19 Nov 2008
Last Updated on STN: 19 Nov 2008

AB Pulmonary fibrosis is a disease characterized by progressive scarring of the lungs, with idiopathic pulmonary fibrosis (IPF) being the most aggressive form. The diagnosis of IPF is made after other conditions are excluded and is based on a characteristic clinical presentation, radiographic features and, sometimes, pathologic specimen. Existing IPF drug regimens, including corticosteroids and cytotoxic medications, are generally ineffective. To date, only lung transplantation has been shown to improve mortality in carefully selected patients. Multiple therapeutic agents have been investigated but none have proven to be successful. Novel drugs are constantly being sought in an attempt to find a therapy that halts or reverses this disease. Imatinib mesylate is used for chronic myelogenous leukemia and gastrointestinal stromal tumors. It also has antifibrotic properties, as demonstrated in several studies using mouse models of pulmonary fibrosis. Currently, trials are underway to investigate its efficacy in human subjects with IPF.

L5 ANSWER 5 OF 10 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2008446413 EMBASE

TITLE: Therapies for interstitial lung disease: Past, present and future.
AUTHOR: Kim, Robert
CORPORATE SOURCE: Lung Transplantation, 600 Highland Avenue, Madison, WI 53792-9988, United States.
AUTHOR: Meyer, Keith C., Dr. (correspondence)
CORPORATE SOURCE: Pulmonary and Critical Care Medicine, Department of Medicine, University of Wisconsin School of Medicine and Public Health, Madison, WI, United States. kcm@medicine.wisc.edu
SOURCE: Therapeutic Advances in Respiratory Disease, (2008) Vol. 2, No. 5, pp. 319-338.
Refs: 138
ISSN: 1753-4658; E-ISSN: 1753-4666
PUBLISHER: SAGE Publications Ltd, 55 City Road, London, EC1Y 1SP, United Kingdom.
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
026 Immunology, Serology and Transplantation
030 Clinical and Experimental Pharmacology
031 Arthritis and Rheumatism
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 30 Sep 2008
Last Updated on STN: 30 Sep 2008

AB As our understanding of the pathobiology and natural history of the various forms of interstitial lung disease (ILD) has evolved, so have our approaches to treating this heterogeneous group of lung disorders. The earliest pharmacologic agents used to treat various forms of ILD were corticosteroids, and corticosteroids are currently the mainstay of therapy for many forms of ILD. However, it has become clear that corticosteroids and other anti-inflammatory agents lack efficacy for many forms of ILD, such as idiopathic pulmonary fibrosis (IPF), and newer therapies that are in clinical trials target the fibrogenic process and/or secondary pulmonary hypertension (PH) that is present in various forms of fibrotic lung disease. Novel therapies, such as the use of biologic agents (antibodies and cell cycle inhibitors) or stem cell therapies will undoubtedly evolve as new research is performed and clinical trials are undertaken. Lung transplantation remains an option for advanced lung disease that is progressive and unresponsive to non-surgical therapies. .COPYRGT. SAGE Publications 2008.

ACCESSION NUMBER: 2008477494 EMBASE
TITLE: [Is there progress in the treatment and comprehension of idiopathic pulmonary fibrosis
?].
Y a-t-il des progres dans le traitement et la comprehension de la fibrose pulmonaire idiopathique?.
AUTHOR: Uzunhan, Y. (correspondence)
CORPORATE SOURCE: Service de Pneumologie, Hopital Avicenne et UPRES 2363, Universite Paris 13, 125, rue de Stalingrad, 93009 Bobigny, France. yurdagul.uzunhan@avc.aphp.fr
AUTHOR: Bonniaud, P.
CORPORATE SOURCE: Service de Pneumologie et Reanimation Respiratoire et INSERM U866, CHU du Bocage et Faculte de Medecine, 21079 Dijon, France. philippe.bonniaud@chu-dijon.fr
SOURCE: Revue des Maladies Respiratoires, (September 2008) Vol. 25,

No. ATS, pp. 79-85.
Refs: 19
ISSN: 0761-8425 CODEN: RMREEY
PUBLISHER: Elsevier Masson SAS, 62 rue Camille Desmoulins, Issy les Moulineaux Cedex, 92442, France.
COUNTRY: France
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 006 Internal Medicine
015 Chest Diseases, Thoracic Surgery and Tuberculosis
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: French
ENTRY DATE: Entered STN: 3 Nov 2008
Last Updated on STN: 3 Nov 2008

L5 ANSWER 7 OF 10 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2008162065 EMBASE
TITLE: New perspectives in the treatment of idiopathic pulmonary fibrosis.
AUTHOR: Rogliani, Paola, Prof. (correspondence)
CORPORATE SOURCE: Policlinico Universitario Tor Vergata, Viale Oxford 81, 00133 Rome, Italy. paola.rogliani@uniroma2.it; saltini@med.uniroma2.it
AUTHOR: Mura, Marco; Assunta Porretta, Maria; Saltini, Cesare
CORPORATE SOURCE: UOC Malattie Respiratorie, Policlinico Tor Vergata, University of Rome Tor Vergata, Rome, Italy. saltini@med.uniroma2.it
SOURCE: Therapeutic Advances in Respiratory Disease, (Sep 2008) Vol. 2, No. 2, pp. 75-93.
Refs: 138
ISSN: 1753-4658; E-ISSN: 1753-4666
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
037 Drug Literature Index
038 Adverse Reactions Titles
005 General Pathology and Pathological Anatomy
FILE SEGMENT: ClinicalTrials.gov
CLINICAL TRIAL NO.: NCT00063869; NCT00125385; NCT00131274; NCT00162760; NCT00359736; NCT00391443; NCT00463983; NCT00514683; NCT00517933; NCT00518310; NCT00532233
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 15 Apr 2008
Last Updated on STN: 15 Apr 2008

AB Idiopathic pulmonary fibrosis (IPF) is the most frequent idiopathic interstitial pneumonia with a prevalence ranging from 5 to 15 per 100,000 persons, and above 175 per 100,000 in the older population. IPF is a relentlessly progressive fibrotic lung disorder leading to death within a median duration of 3 years. It was hypothesized in the 1970s that pulmonary fibrosis initiates as an "alveolitis" progressing to interstitial fibrosis with connective tissue deposition, derangement of the lung architecture and functional impairment. However, in vitro studies indicated that alveolar/bronchiolar injured epithelial cells can drive the fibrotic process in the absence of macrophages and with minimal inflammation. This, together with the inability of classic immunosuppressive therapy to cure IPF, generated new pathogenesis paradigms and intense research into the role of the lack or the excessive production of anti-fibrotic or profibrotic mediators, oxidant injury, exaggerated coagulation, thus leading to investigate new treatment

strategies. Preliminary results of some of such trials have shown significant reductions in lung function decline, disease exacerbation and mortality. .COPYRGT. Sage Publications 2008.

L5 ANSWER 8 OF 10 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2007365137 MEDLINE
DOCUMENT NUMBER: PubMed ID: 17579094
TITLE: Thalidomide prevents bleomycin-induced pulmonary fibrosis in mice.
AUTHOR: Tabata Chiharu; Tabata Rie; Kadokawa Yoshio; Hisamori Shigeo; Takahashi Meiko; Mishima Michiaki; Nakano Takashi; Kubo Hajime
CORPORATE SOURCE: Horizontal Medical Research Organization, Graduate School of Medicine, Kyoto University, Kyoto, Japan.. ctabata@hyo-med.ac.jp
SOURCE: Journal of immunology (Baltimore, Md. : 1950), (2007 Jul 1) Vol. 179, No. 1, pp. 708-14. Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200708
ENTRY DATE: Entered STN: 21 Jun 2007
Last Updated on STN: 8 Aug 2007
Entered Medline: 7 Aug 2007

AB Pulmonary fibrosis in humans can occur as a result of a large number of conditions. In idiopathic pulmonary fibrosis (IPF), pulmonary function becomes progressively compromised resulting in a high mortality rate. Currently there are no proven effective treatments for IPF. We have recently reported that IL-6 and TGF-beta(1) plays an important role in proliferation and differentiation of lung fibroblasts, and all-trans-retinoic acid (ATRA) prevented bleomycin-induced lung fibrosis through the inhibition of these cytokines. Thalidomide (Thal) has been used in the treatment of multiple myeloma through the inhibitory effect on IL-6-dependent cell growth and angiogenesis. In this study, we examined the preventive effect of Thal on bleomycin-induced pulmonary fibrosis in mice. We performed histological examinations and quantitative measurements of IL-6, TGF-beta(1), collagen type Ialpha1 (COL1A1), vascular endothelial growth factor (VEGF), angiopoietin-1 (Ang-1) and angiopoietin-2 (Ang-2) in bleomycin-treated mouse lung tissues with or without the administration of Thal. Thal histologically ameliorated bleomycin-induced fibrosis in mouse lung tissues. Thal decreased the expressions of IL-6, TGF-beta(1), VEGF, Ang-1 Ang-2, and COL1A1 mRNA in mouse lung tissues. In addition, Thal inhibited angiogenesis in the lung. In vitro studies disclosed that Thal reduced 1) production of IL-6, TGF-beta(1), VEGF, Ang-1, and collagen synthesis from human lung fibroblasts, and 2) both IL-6-dependent proliferation and TGF-beta(1)-dependent transdifferentiation of the cells, which could be the mechanism underlying the preventive effect of Thal on pulmonary fibrosis. These data may provide a rationale to explore clinical use of Thal for the prevention of pulmonary fibrosis.

L5 ANSWER 9 OF 10 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2006581368 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16837501
TITLE: Thalidomide reduces IL-18, IL-8 and TNF-alpha release from alveolar macrophages in interstitial lung disease.
AUTHOR: Ye Q; Chen B; Tong Z; Nakamura S; Sarria R; Costabel U; Guzman J
CORPORATE SOURCE: Dept of Pneumology and Allergology, Ruhrlandklinik, Medical

Faculty, University of Essen, Essen, Germany.
SOURCE: The European respiratory journal : official journal of the European Society for Clinical Respiratory Physiology, (2006 Oct) Vol. 28, No. 4, pp. 824-31. Electronic Publication: 2006-07-12.
Journal code: 8803460. ISSN: 0903-1936.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200702
ENTRY DATE: Entered STN: 3 Oct 2006
Last Updated on STN: 2 Feb 2007
Entered Medline: 1 Feb 2007

AB Thalidomide exhibits diverse actions of anti-inflammation, immunomodulation and anti-angiogenesis. The efficacy of thalidomide treatment in sarcoidosis with lupus pernio is thought to be due to inhibition of tumour necrosis factor (TNF)-alpha. The mechanisms that underlie the properties of thalidomide are still unclear in interstitial lung disease. The current authors investigated the potential inhibitory effects of thalidomide at concentrations of 0.1, 0.01 and 0.001 mM on the production of transforming growth factor-beta, TNF-alpha, interleukin (IL)-1beta, IL-6, IL-8, IL-10, IL-12p70, IL-12p40 and IL-18 by alveolar macrophages from bronchoalveolar lavage in patients with sarcoidosis (n = 8), hypersensitivity pneumonitis (HP; n = 8) and idiopathic pulmonary fibrosis (IPF; n = 12). In sarcoidosis and HP patients, thalidomide induced a dose-dependent, partial suppression of lipopolysacchride (LPS)-stimulated TNF-alpha, IL-12p40 and IL-18 release. At the highest thalidomide concentration (0.1 mM), LPS-stimulated IL-8 production was also suppressed. In IPF patients, although spontaneous production of TNF-alpha, IL-12p40, IL-18 and IL-8 was lower than in sarcoidosis and HP patients, with LPS stimulation the cytokines were significantly elevated and also partially inhibited by thalidomide. In conclusion, thalidomide has the potential to improve the therapeutic regimens for sarcoidosis, hypersensitivity pneumonitis and idiopathic pulmonary fibrosis by reducing tumour necrosis factor-alpha, interleukin-12p40, interleukin-18 and interleukin-8 production.

L5 ANSWER 10 OF 10 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2006085203 EMBASE
TITLE: [New treatments for idiopathic pulmonary fibrosis].
Nuevos tratamientos en la fibrosis pulmonar idiopatica.
AUTHOR: Montero-Martinez, C., Dr. (correspondence)
CORPORATE SOURCE: Servicio de Neumologia, Hospital Universitario Juan Canalejo, A Coruna, Spain. carmen_montero@canalejo.org
AUTHOR: Montero-Martinez, C., Dr. (correspondence)
CORPORATE SOURCE: Servicio de Neumologia, Complejo Hospitalario Universitario Juan Canalejo, Xubias de Abajo, 84, 15006 A Coruna, Spain. carmen_montero@canalejo.org
SOURCE: Archivos de Bronconeumologia, (Jan 2006) Vol. 42, No. 1, pp. 1-2.
Refs: 18
ISSN: 0300-2896 CODEN: ARBRDA
COUNTRY: Spain
DOCUMENT TYPE: Journal; Editorial
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
037 Drug Literature Index
005 General Pathology and Pathological Anatomy

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                    009     Surgery
LANGUAGE:           Spanish; Castilian
ENTRY DATE:         Entered STN: 10 Mar 2006
                    Last Updated on STN: 10 Mar 2006


=> d his

     (FILE 'HOME' ENTERED AT 15:41:36 ON 23 OCT 2009)

     FILE 'REGISTRY' ENTERED AT 15:41:47 ON 23 OCT 2009
L1            29 S THALIDOMIDE

     FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 15:42:01 ON 23 OCT 2009
L2         20958 S L1
L3          6721 S "IDIOPATHIC PULMONARY FIBROSIS"
L4            15 S L2 AND L3
L5            10 DUP REM L4 (5 DUPLICATES REMOVED)

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COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                     ENTRY    SESSION
FULL ESTIMATED COST                                  34.66      40.71

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FILE LAST UPDATED: 22 Oct 2009  (20091022/ED)
REVISED CLASS FIELDS (/NCL) LAST RELOADED:  Aug 2009
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE:  Aug 2009

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reclassification data for the third quarter of 2009.

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substance identification.

=> s l1
L6          3482 L1

=> s "idiopathic pulmonary fibrosis"
          14708 "IDIOPATHIC"
              2 "IDIOPATHICS"
          14708 "IDIOPATHIC"
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                ("IDIOPATHIC" OR "IDIOPATHICS")
         113751 "PULMONARY"
              2 "PULMONARIES"
         113751 "PULMONARY"
                ("PULMONARY" OR "PULMONARIES")
          50853 "FIBROSIS"
              1 "FIBROSISES"
          50853 "FIBROSIS"
                ("FIBROSIS" OR "FIBROSISES")
L7          897 "IDIOPATHIC PULMONARY FIBROSIS"
                ("IDIOPATHIC"(W)"PULMONARY"(W)"FIBROSIS")

=> s l6 and l7
L8            2 L6 AND L7

=> d l8 1-2 ibib abs

L8   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER:         2007:659399  CAPLUS
DOCUMENT NUMBER:          147:63664
TITLE:                    Thalidomide Prevents Bleomycin-Induced Pulmonary
                          Fibrosis in Mice
AUTHOR(S):                Tabata, Chiharu; Tabata, Rie; Kadokawa, Yoshio;
                          Hisamori, Shigeo; Takahashi, Meiko; Mishima, Michiaki;
                          Nakano, Takashi; Kubo, Hajime
CORPORATE SOURCE:         Horizontal Medical Research Organization, Graduate
                          School of Medicine, Kyoto University, Kyoto, Japan
SOURCE:                   Journal of Immunology (2007), 179(1), 708-714
                          CODEN: JOIMA3; ISSN: 0022-1767
PUBLISHER:                American Association of Immunologists
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   Pulmonary fibrosis in humans can occur as a result of a large number of
     conditions.  In idiopathic pulmonary fibrosis
     (IPF), pulmonary function becomes progressively compromised resulting in a
     high mortality rate.  Currently there are no proven effective treatments
     for IPF.  We have recently reported that IL-6 and TGF-β1 plays an
     important role in proliferation and differentiation of lung fibroblasts,
     and all-trans-retinoic acid (ATRA) prevented bleomycin-induced lung
     fibrosis through the inhibition of these cytokines.  Thalidomide (Thal)
     has been used in the treatment of multiple myeloma through the inhibitory
     effect on IL-6-dependent cell growth and angiogenesis.  In this study, we
     examined the preventive effect of Thal on bleomycin-induced pulmonary
     fibrosis in mice.  We performed histol. examns. and quant. measurements of
     IL-6, TGF-β1, collagen type Iα1 (COL1A1), vascular endothelial
     growth factor (VEGF), angiopoietin-1 (Ang-1) and angiopoietin-2 (Ang-2) in
     bleomycin-treated mouse lung tissues with or without the administration of
     Thal.  Thal histol. ameliorated bleomycin-induced fibrosis in mouse lung
     tissues.  Thal decreased the expressions of IL-6, TGF-β1, VEGF, Ang-1
     Ang-2, and COL1A1 mRNA in mouse lung tissues.  In addition, Thal inhibited
     angiogenesis in the lung.  In vitro studies disclosed that Thal reduced
     (1) production of IL-6, TGF-β1, VEGF, Ang-1, and collagen synthesis from
     human lung fibroblasts, and (2) both IL-6-dependent proliferation and
     TGF-β1-dependent transdifferentiation of the cells, which could be
     the mechanism underlying the preventive effect of Thal on pulmonary
     fibrosis.  These data may provide a rationale to explore clin. use of Thal
     for the prevention of pulmonary fibrosis.
OS.CITING REF COUNT:      8     THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD
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REFERENCE COUNT:          55    THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS
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L8 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2006:1198847 CAPLUS
DOCUMENT NUMBER: 146:55192
TITLE: Thalidomide reduces IL-18, IL-8 and TNF-α release from alveolar macrophages in interstitial lung disease
AUTHOR(S): Ye, Q.; Chen, B.; Tong, Z.; Nakamura, S.; Sarria, R.; Costabel, U.; Guzman, J.
CORPORATE SOURCE: Dept of Pneumology and Allergology, Ruhrlandklinik, Medical Faculty, University of Essen, Essen, Germany
SOURCE: European Respiratory Journal (2006), 28(4), 824-831
CODEN: ERJOEI; ISSN: 0903-1936
PUBLISHER: European Respiratory Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Thalidomide exhibits diverse actions of anti-inflammation, immunomodulation and anti-angiogenesis. The efficacy of thalidomide treatment in sarcoidosis with lupus pernio is thought to be due to inhibition of tumor necrosis factor (TNF)-α. The mechanisms that underlie the properties of thalidomide are still unclear in interstitial lung disease. The current authors investigated the potential inhibitory effects of thalidomide at concns. of 0.1, 0.01 and 0.001 mM on the production of transforming growth factor-β, TNF-α, interleukin (IL)-1β, IL-6, IL-8, IL-10, IL-12p70, IL-12p40 and IL-18 by alveolar macrophages from bronchoalveolar lavage in patients with sarcoidosis (n = 8), hypersensitivity pneumonitis (HP; n = 8) and idiopathic pulmonary fibrosis (IPF; n = 12). In sarcoidosis and HP patients, thalidomide induced a dose-dependent, partial suppression of lipopolysaccharide (LPS)-stimulated TNF-α, IL-12p40 and IL-18 release. At the highest thalidomide concentration (0.1 mM), LPS-stimulated IL-8 production was also suppressed. In IPF patients, although spontaneous production of TNF-α, IL-12p40, IL-18 and IL-8 was lower than in sarcoidosis and HP patients, with LPS stimulation the cytokines were significantly elevated and also partially inhibited by thalidomide. In conclusion, thalidomide has the potential to improve the therapeutic regimens for sarcoidosis, hypersensitivity pneumonitis and idiopathic pulmonary fibrosis by reducing tumor necrosis factor-α, interleukin-12p40, interleukin-18 and interleukin-8 production
OS.CITING REF COUNT: 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)
REFERENCE COUNT: 37 THERE ARE 37 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 15:41:36 ON 23 OCT 2009)

FILE 'REGISTRY' ENTERED AT 15:41:47 ON 23 OCT 2009
L1 29 S THALIDOMIDE

FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 15:42:01 ON 23 OCT 2009
L2 20958 S L1
L3 6721 S "IDIOPATHIC PULMONARY FIBROSIS"
L4 15 S L2 AND L3
L5 10 DUP REM L4 (5 DUPLICATES REMOVED)

FILE 'CAPLUS' ENTERED AT 15:43:41 ON 23 OCT 2009
L6 3482 S L1
L7 897 S "IDIOPATHIC PULMONARY FIBROSIS"

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L8             2 S L6 AND L7

=>

---Logging off of STN---


=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                      13.22      53.93

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                         ENTRY    SESSION
CA SUBSCRIBER PRICE                                      -1.64      -1.64

STN INTERNATIONAL LOGOFF AT 15:44:29 ON 23 OCT 2009
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